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The President, DR. HENRY R. STEDMAN, in the Chair.

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The Subdivision of the Representation of Cutaneous and Muscular Sensibility and of Stereognosis in the Cerebral Cortex.—By Dr. Charles K. Mills and Dr. T. H. Weisenburg. (See this journal, page 617.)

Dr. Morton Prince said that Dr. Mills and Dr. Weisenburg's communication was not only interesting, but important, both for the evidence they present for the subdivision of the cortical area for the stereognostic sense, and for the localization of this sense as a whole. These are two different problems.

The cases of Dr. Mills and Dr. Weisenburg, we are told, are the only ones on record with the limitation of this faculty to a part of the hand. Dr. Prince would not add any personal experience showing this limitation. He had, however, a case of astereognosis from tumor which he would like to take this opportunity of putting on record along side of Dr. Mills and Dr. Weisenburg's cases, as it bears on the second problem of localization of stereognosis and on the segmental representation of common sensibility in the cortex. The epileptiform attacks of pain, spreading from segment to segment of the body like motor spasms, are also of interest.

First, Dr. Prince merely suggested that logically stereognosis may well have a divisional representation; just as it is a function entirely confined to the hand, and does not exist, excepting possibly in a very primitive manner, in the foot or body. We may, of course, be able to recognize objects with the foot or toes, and an ape probably does, but this is by a logical process of inference or guessing rather than by a distinct stereognostic perception. If common sensation has segmental representation in the cortex, as Dr. Mills has insisted, as Campbell from his histological researches has strongly argued, and, as Dr. Prince supposed, has been maintained all along by those who believe in the central convolutions being sensori-motor, then we should expect that the stereognostic sense, theoretically depending as it does on the primary sensations, would be similarly represented. This view particularly harmonizes with Campbell's researches by which he has endeavored on histological and other grounds, to show that the postcentral gyrus is not only the seat of common sensation, but, in its posterior part, of the muscular sense and stereognosis. In amputation of the limbs, it will be remembered that he found changes segmentally focussed in this convolution, corresponding in location with those he found in the anterior motor convolution where they were limited to the centers for the amputated movements.

This histological evidence is not of course proof; it is only suggestive, but harmonizes with the view which Dr. Mills and Dr. Weisenburg advance on the strength of their cases.

The case which Dr. Prince reported,[1] so far as it goes, supports Campbell's contention for the location of the muscular and stereognostic senses in the postparietal gyrus as maintained by Dr. Mills. The interesting points of the case are: The limitation of the area of tactile anesthesia (from a cortical lesion) to a circumscribed region over the neck and face; the peculiar epileptiform diffusion of pain; the loss of muscular sense, and the astereognosis and muscular atrophy associated with a tumor, probably primarily located in the posterior central convolution, although eventually involving both central convolutions.

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[1] The case will be reported in full in this journal.

Yet it should not be overlooked that it is not possible to say that, through pressure, the tumor did not affect the parietal region.

Whether the complex function of stereognosis is to be located in the hinder portion of the postcentral convolution, as Campbell believes, or farther back in the posterior portion of the superior parietal, as Oppenheim and Mills hold, or between the two, is, in Dr. Prince's opinion, far from certain, and we are not entitled to speak dogmatically or categorically in the matter. The cases which Mills,[2] for example, cites in support of his contention in his recent paper, are open to two or more interpretations, particularly in the light of the new theory that the postcentral gyrus is sensory, and the motor area is anterior to the fissure of Rolando. An analysis of these cases shows:

(1) The symptoms in Oppenheim's case are explicable on the theory of pressure upon the postcentral convolution; which must have existed in fact, as there was paralysis, showing that pressure was exerted upon the precentral, and therefore *through* the postcentral.

(2) In one of the cases of Dr. Mills and Dr. Weisenburg there was also *hemiparesis,* and, as they report, the necropsy revealed a lesion which encroached upon the postcentral and precentral convolutions, diffuse meningitis being also present. Secondary degenerations due to these cortical lesions were also traced.

In another of Dr. Mills and Dr. Weisenburg's cases, in the absence of an autopsy, it is equally possible that the postcentral convolution was involved.

(3) In another case there was also *paresis* with early ankle clonus; and, although the tumor was found by Keen in the parietal region, it is possible that the astereognosis was caused—as, indeed, Mills must on his own views explain the loss of muscular sense—by pressure on the postcentral region or its neighborhood. How else explain the loss of muscular sense on his theory of its localization? and if the muscular sense, why not the astereognosis?

(4) In another case there was also *paresis* with increased reflexes, ankle clonus and Babinski. The loss of muscular sense and the astereognosis are therefore open to the same interpretation, though the tumor was found by Keen to be located *"in the main"* in the superior parietal gyrus.

(5) In another case the lesion was deep and invaded the subcortex of the postcentral, as well as that of the parietal region.

(6) Another case was also a *"subcortical postfrontal,"* as well as parietal growth.

(7) Another case was a large parietal tumor with spastic hemiplegia (probably involving the internal capsule). It was not determined whether there were astereognosis and loss of muscular sense.

Dr. Spiller's[3] three cases, analyzed, show the following: In one there was temporary hemiplegia which, clearing up, left an ataxia. Sensation could not be determined on account of the mental condition. At the autopsy there was a hemorrhagic area "about two centimeters or three centimeters" behind the Rolandic fissure—pretty close to eliminate involvement of the postcentral fissure, particularly as there had been hemiplegia.

In the second case there was astereognosis, loss of sense of position and impaired sensation for touch, but Dr. Spiller says that the postcentral convolution was involved in its extreme upper part by a tuberculous plaque.

In the third case there was paresis as well as ataxia and astereognosis, showing that the softening of the parietal lobe must have gone as far forward as the central fissure.

Astereognosis may have its seat in the posterior portion of the superior parietal lobe, but it is not on this kind of evidence that we are entitled to speak dogmatically. There may be other cases more to the point which are harder to meet, but Dr. Prince had not attempted to go through the whole literature of the subject.

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[2] University of Pennsylvania Medical Bulletin, April-May, 1906.

[3] American Journal of the Medical Sciences, February, 1904.

In his judgment it is still an open question whether stereognosis has its location in the postcentral, or in the neighborhood of the postcentral, or farther back in the parietal lobe.

Dr. Walton agreed with Dr. Mills regarding the separation of the different varieties of sensory representation and the separation of the cortex into definite regions. He did not, however, feel that Dr. Mills has quite established the seat of the stereognostic sense in the parietal region. Dr. Walton would like to remind the members of the theory suggested by Dr. Paul and himself a few years ago. Their idea was that just as kinesthetic speech memories are placed near the motor centers for larynx and tongue, just so the stereognostic sense which immediately precedes movement of the arm has its seat in close proximity to, but still separated from, the motor arm center. They reported several cases of postcentral lesion in which the stereognostic sense was lost, while tactile sense was retained; they had looked in vain for reports of similar dissociation in cases of parietal lesion. In all the cases reported by Dr. Mills and Dr. Weisenburg the association of astereognosis with loss of tastile sense is noteworthy.

Dr. Spiller referred to the pain described by Dr. Prince in his patient independent of the convulsive attacks. The pain occurred in the hand, extended up the arm and into the shoulder. He desired Dr. Prince to mention, if possible, whether the pain was confined to the ulnar side at the beginning of the attack, or whether the whole hand was involved. In the case of astereognosis from syphilis of the parietal lobe reported by Dr. Mills and Dr. Weisenburg, pain of one upper limb had been a persistent symptom. The patient had been under his (Dr. Spiller's) care off and on many years, and nothing seemed to relieve the pain in the limb. Pain from a cortical or slightly subcortical lesion is an interesting phenomenon. Dr. Spiller agreed with Dr. Prince in that he believed that at the present time we cannot separate the postcentral convolution from the parietal in their functional manifestations.

Dr. Knapp said in the first place he was very glad that Dr. Mills had at last recognized the case he (Dr. Knapp) reported some years ago, which he contested at the time. In the second place, to come back to the old question of stereognostic conception, he did not believe that cases of astereognosis have been sufficiently carefully analyzed to enable us to determine one particular point. We know, for example, in the more highly differentiated forms of auditory agnosis an apparent agnosis may be due to simple failure of sensory perception, as for instance, when we fail to recognize spoken language through the closed door, or fail to recognize conversation in a low tone. We must bear in mind that many of the forms of sensibility, particularly those forms of highly educated muscular sense as found in the fencer or expert billiard player, are beyond our methods of research; yet there may be so much blunting of perception that the special recognition of stereognosis may be affected. He did not think these cases have been sufficiently analyzed as yet to know whether there is true astereognosis or simply sensory impairment.

In regard to these definite areas, he felt somewhat skeptical. There is a process of education whereby the hand becomes the most highly educated part of our body, and the thumb and forefinger more than the other fingers. Clinically we find, for example, that in the ordinary cases of hemiplegia the blunting of sensibility is always more marked in the distal portion of the limb, gradually fading away, whereas in the hysterical cases and sometimes in organic cases, as in a case he showed a few years ago at the meeting of the Boston Society of Pschiatry and Neurology, we get the sharply defined anesthesia, but in the ordinary cerebral cases we get one which gradually fades away. That, he thought, is due to the fact of its being a process of education, and the acquired forms of decay disappear in the most highly educated parts first.

Dr. Langdon said in regard to the subject of division and subdivision of the cortical areas that some years ago he reported the case of a man where the skull had been fractured in the post-parietal region, and the patient's stereognostic sense was affected. He could recognize some objects, but when he held a coin in his right hand he could only recognize it as a half-moon or half-disk. The experiment was tried again and again, but he persisted in the statement that he could recognize the coin as a half circle not as a disk.

Dr. Charles L. Dana thought this discussion ought not to be closed without speaking of the work of Dr. Campbell. It seems to him (Dr. Dana) that his work in this connection has gone a long way to determine the anatomy and functions of these regions.

Personally, he was not sure that the precentral convolution is the only motor area. At least he thought it is not absolutely demonstrated as yet. We should not forget that the anterior and posterior convolutions are very closely connected anatomically and physiologically. The posterior convolution is no doubt mainly a sensory area and may seem to consist of a different kind of cells than the anterior, but there is a very intimate association between the two convolutions. Dr. Sherrington told Dr. Dana that in his experiments he had found that when he stimulated a posterior central convolution (this part first) he did not get any reaction, but if he stimulated the anterior convolution first and then the posterior he got a motor reaction then. In other words, he did get movements from stimulation of the posterior convolution.

So far as the differentiation of the sensory areas is concerned, Dr. Dana was quite ready to agree with the general view of Dr. Mills. The only point he thought we might question is whether he has yet proved his special subdivisions.

One point more in regard to astereognosis. He agreed entirely with Dr. Mills that it is a co-ordinating faculty of the mind rather than a sensory process. Its loss is like that occurring in an aphasia. This view he advocated many years ago.

Dr. Weisenburg said that the pains complained of by Dr. Prince's patient were similar to those of case 1. Dr. Weisenburg believed that it was possible to have central pain due to an interruption of the sensory tracts anywhere in their course, and that he had cases proving this. He admitted Dr. Walton's criticism that there was always some motor involvement in the supposedly pure sensory cases, but thought this could be explained in the same manner as the paresis of the seventh nerve in involvements of the sensory fifth.

Dr. Mills said in closing the discussion:

He believed that the motor area was in front of the central fissure, and was one of the first to assert that it was so placed, although at first he did not put it entirely there.

With regard to Dr. Knapp he had never questioned his facts, nor his manner of presenting them, although he had sometimes his inferences. His case published in 1891 was clearly in favor of sensorial subdivisional representation.

Dr. Walton is simply getting the cart before the horse in his location of the sensory and stereognostic areas in relation to the motor zone.

With regard to the separateness of stereognostic conception and forms of sensation, it might be remarked that there was at the Philadelphia Hospital a case in which, although all the forms of sensibility were completely lost, the patient, if given sufficient time, was able to recognize objects by manipulation, the eyes of course being blind-folded.

With regard to Dr. Dana it should be said that he was one of the first to recognize the fact that the cortical representation of sensation like that of motion was subdivided; in other words, that there were sensory areas and centers for the limbs, face and portions of these. Dr. Dana, however, like Horsley and others, held to the view that these subdivided sensory and motor areas were practically identical.

With regard to the work of Campbell, this had been fully recognized by Dr. Mills, as had also that of Grünbaum and Sherrington. Some of the experiments of the latter in particular would seem to point to centers in the sensory cortex correlated with special motor centers.

(*To be continued.*)